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**Medical Faculty**  
**Department of Internal Medicine**

# **CLINICAL CASE**

## **COMPLICATED**

## **MIOCARDIAL INFARCTION**

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# Introduction

- The clinical course of myocardial infarction frequently burdened by a variety of complications, which largely determine its course and prognosis
- Complications of acute MI are different and life threatening
- If a person survives the sudden loss by ischemic necrosis of part of the ventricular muscle there follows a period in which special dangers threaten each of the physical systems and the personality itself
- These dangers arise from two principal sources; on the one hand, from the local effects of the lesion and the circulatory depression that follows; and, on the other, from the hazards that may attend during active treatment
- The onset of each of these complications usually results in explicit symptoms and physical manifestations; thus, a basic knowledge of the complications that occur in the postinfarction period and the clinical syndromes associated with each, will allow the physician to evaluate and treat the complication appropriately
- Prompt diagnosis and therapy are essential

# Goal



The purpose of this presentation is to introduce to the complications that may arise in the course of acute MI and demonstrate that various therapeutic modalities, both medical and surgical, should be able to improve not only symptoms but also survival of the patient

# Definition



**Acute myocardial infarction (MI)** defines cardiomyocyte necrosis in a clinical setting consistent with acute myocardial ischaemia

# Complications Of MI

## CLASSIFICATION

- Mechanical
- Arrhythmic
- Ischemic
- Inflammatory
- Embolic

# Mechanical Complications of MI

## CLASSIFICATION

- Left ventricular failure and cardiogenic shock
- Ventricular free wall rupture (VFWR)
- Ventricular septal rupture (VSR)
- Papillary muscle rupture with severe mitral regurgitation (MR)
- Ventricular aneurysm

## RISK FACTORS

- age older than 60 years
- female gender
- pre-existing hypertension
- lack of left ventricular hypertrophy
- and no previous MI (pre-existing scarring tends to prevent myocardial tearing).

# Mechanical Complications of MI

## Ventricular Rupture



### PATOPHYSIOLOGY

Rupture can occur at almost any time after MI but is most common 3 to 7 days after infarction. It is at this point in the healing process that lysis of the myocardial connective tissue is maximal and the granulation tissue has not deposited sufficient collagenous matrix to buttress the wall.

# Mechanical Complications of MI

## Left Ventricular Aneurysm

Left ventricular aneurysm (LVA) is defined as a localized area of myocardium with abnormal outward bulging and deformation during both systole and diastole.

### **RISK FACTORS**

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- female sex
- total occlusion of the LAD artery
- single-vessel disease
- absence of previous angina.



# Mechanical Complications of MI

## Left Ventricular Aneurysm



### PATHOPHYSIOLOGY

A late complication, most commonly result from a large transmural anteroseptal infarct that heals with the formation of thin scar tissue.

Complications of ventricular aneurysms include mural thrombus, arrhythmias, and heart failure, but rupture of the fibrotic wall does not occur

# Mechanical Complications of MI

## Left Ventricular Failure and Cardiogenic Shock

- Left ventricular failure (pump failure) is defined as the condition in which cardiac output is insufficient to perfuse various body organs because of acute left ventricular contractile dysfunction caused by myocardial infarction
- Cardiogenic shock clinically is defined as:
  - Sustained systolic BP < 90 mmHg without hypovolemia, in combination with signs of organ severe, prolonged tissue hypoperfusion (e.g. oliguria, impaired consciousness) and signs of sympathetic activation (e.g. cool extremities, sweating)

# Mechanical Complications of MI

## Left Ventricular Failure and Cardiogenic Shock

### PATOPHYSIOLOGY

- LV dysfunction results in low stroke volume reducing cardiac output. Myocardial perfusion is compromised by systemic hypotension and increased LV end-diastolic pressure
- Activation of compensatory mechanisms:
  - Tachycardia (increases myocardial oxygen demand)
  - Increased myocardial contractility in nondiseased myocardial segments
  - Venoconstriction to increase effective circulating blood volume
  - Neurohormonal (RAAS, vasopressin) activation lead to peripheral arterial vasoconstriction; although increasing peripheral resistance increases blood pressure, it also reduces tissue perfusion

# Arrhythmic Complications of MI

## CLASSIFICATION

- Supraventricular tachyarrhythmias: sinus tachycardia, premature atrial contractions, paroxysmal supraventricular tachycardia, atrial flutter, and atrial fibrillation
- Accelerated junctional rhythms
- Bradyarrhythmias: sinus bradycardia and junctional bradycardia
- Atrioventricular (AV) blocks: first-degree AV block, second-degree AV block, and third-degree AV block
- Intraventricular blocks: left anterior fascicular block, right bundle branch block (RBBB), and left bundle branch block (LBBB)
- Ventricular arrhythmias: premature ventricular contractions (PVCs), accelerated idioventricular rhythm, ventricular tachycardia, and ventricular fibrillation
- Reperfusion arrhythmias

# Arrhythmic Complications of MI

## PATOPHYSIOLOGY

- AMI is characterized by generalized autonomic dysfunction that results in enhanced automaticity of the myocardium and conduction system. Electrolyte imbalances (eg, hypokalemia and hypomagnesemia) and hypoxia further contribute to the development of cardiac arrhythmia. The damaged myocardium acts as substrate for re-entrant circuits, due to changes in tissue refractoriness.
- Enhanced efferent sympathetic activity, increased concentrations of circulating catecholamines, and local release of catecholamines from nerve endings in the heart muscle itself have been proposed to play roles in the development of peri-infarction arrhythmias. Furthermore, transmural infarction can interrupt afferent and efferent limbs of the sympathetic nervous system that innervates myocardium distal to the area of infarction. The net result of this autonomic imbalance is the promotion of arrhythmias.

# Ischemic Complications of MI

## Recurrent ischemia/reinfarction



- Recurrent postinfarction angina may be due to either an infarct extension or reinfarction in a separate territory or reocclusion of the infarction related artery
- Postinfarction angina is important because it increases morbidity and mortality

# Inflammatory Complications of MI

## CLASSIFICATION

- Early pericarditis
- Late pericarditis (Dressler's syndrome)

# Inflammatory Complications of MI

## Early Pericarditis



### PATHOPHYSIOLOGY

- Usually develops within 24-96 after AMI.
- Pericarditis (fibrinous or hemorrhagic) is caused by inflammation of pericardial tissue overlying infarcted myocardium.



# Inflammatory Complications of MI

## Post-MI Syndrome (Dressler syndrome)



### PATHOPHYSIOLOGY

- Symptoms of pericarditis occurring 2-3 weeks after AMI
- The exact mechanism has yet to be elucidated, post-MI syndrome is considered to be an autoimmune process.

# Embolism

## Left ventricular mural thrombus

Frequently develops after anterior infarcts of the LV wall.

LVMT is associated with a high risk of systemic embolization (the most common clinical presentation is stroke ).

### **RISK FACTORS**

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- LV regional-wall akinesia or dyskinesia with blood stasis
- injury to and inflammation of the endocardial tissue that provides a thrombogenic surface
- a hypercoagulable state.

# Embolic

## Left ventricular mural thrombus



### PATHOPHYSIOLOGY

With any infarct, the combination of a local loss of contractility (causing stasis) with endocardial damage (causing a thrombogenic surface) can foster mural thrombosis



PATIENT K.

65 y.o.

# Complains

- Burning central chest pain, more than 60 min duration, without any radiation, nitroglycerine intake doesn't relief pain, abrupt onset, severity 7
- Dyspnea at rest, exacerbated by minimal physical exertion

# Present illness

- Central chest pain and worsening symptoms of dyspnea had been appeared abruptly, when the patient was at home and carried out household chores
- Patient took nitroglycerine three times, but it did not relief pain
- After 30 min patient had called to emergency. ECG had been recorded. Signs of STEMI of the posterior wall had been found and patient had been delivered to cardiologic emergency department

# Past medical history

- Over 20 years patient suffer from essential hypertension. Patient said that prescribed by cardiologist medications (ACE inhibitors, diuretics and b-blockers) had been taken regularly, but BP level had not been controlled properly (it occurred rising of BP to 160-200/100 mm Hg)
- 1999 year: Carotid artery atherosclerosis, left internal carotid endarterectomy
- Since 2003 year bother persistent atrial fibrillation, tachysystolic form, which was successfully converted to sinus rhythm by pharmacologic cardioversion (IV amiodarone)
- 25.02.2010 STEMI inferior wall complicated by cardiogenic shock, Dressler's syndrome
- 06.12.2010 Ischemic stroke in the circle of Willis

# Drug history



- Nebivalol 5 mg pd
- Losartan 50 mg pd
- Hydrochlorothiazide 25 mg pd
- Aspirin 75 mg pd
- Clopidogrel 75 mg pd
- Atorvastatin 20 mg pd



# Allergies and reactions



- Patient has not any allergies
- Patient has not any reactions to drugs and medication

# Alcohol and Smoking

- Alcohol consumption 1,5 L of normal beer per day = 42 units of alcohol per week
- Smoking 1,5-2 packs per day during 40 years = 60-80 pack-years

# Family history



- Patient's mother and brother suffer from essential hypertension

## VITAL SIGNS:

Temperature	– 36,8° C
PS	– 40 bpm
BP	– 110/60 mm Hg
Respiratory rate	– 17 pm
High	– 188 cm
Weight	– 105 kg
BMI	– 30,2 kg/m <sup>2</sup>

# Inspection | 2

- Elderly male, has correct **orientation** in space and surroundings, mild depressed
- **The posture** is orthopnea (the patient uses 3 pillows)
- **Skin** is pale, mild cyanosis of the lips, fingers and toes, rashes and hemorrhages are absent
- **Turgor and elasticity** of the skin is decreased
- **Subcutaneous fat tissue** is increased, predominantly in abdominal zone (central obesity, waist circumflex 138 cm)
- **Nails** are without any abnormalities
- **Mucous membranes** are pale and wet
- **Tongue** is clear and wet
- **Severe oedema** of the low extremities (3+)
- **Lymph nodes** are not palpable
- **Joints** are normal, active and passive movements are painless

- **The head and neck** examination is normal
- **Carotid pulsation.** JVP 6,5 cm above the sternal angle
- **The chest** is normal shape
- Decrease breath sounds and bibasilar coarse crackles of the lungs to **auscultation**
- **The point of apex** beat is diffuse, 3 cm in diameter, shifted to the left (palpated 1,5-2 cm to the left from medclavicular line in the 5<sup>th</sup> intercostal space)
- **S1** and **S2** are soft. **Holosystolic murmur** best heard at the tricuspid valve
- **Abdomen** is soft and nontender. Hepatomegaly (+ 4 cm), **liver** palpation is tender. **The kidneys** are not palpable
- **Stool** is normal
- **Urination** is normal



# Preliminary diagnosis

Coronary artery disease. Repeated myocardial infarction (28.08.2015). Postinfarction inferior wall cardiosclerosis (2006). Killip IV

Essential hypertension III stage, 2 grade

Persistent atrial fibrillation, tachysystolic form

# Plan of survey

## **BASIC TESTS:**

- Complete blood count
- Urinalysis
- Biochemical blood profile  
(Glucose, Bilirubin, ALT, AST, Creatinine, Urea, Potassium, Total protein Cardiac Biomarkers)
- ECG
- Echocardiography
- Chest X-Ray

## **ADDITIONAL RECOMMENDATIONS:**

- Blood lipid profile
- Coagulogram
- B-NP
- Coronary angiography



## Complete blood count

28.08.2015

ESR 3 mm/h

WBC (N 4,0-9,0 $10^9/L$ )	11,8 $10^9/L$	
NE (N 1,7-7,7 $10^9/L$ ; 47,0-72% )	9,1 $10^9/L$	77,8%
LY (N 0,4-4,4 $10^9/L$ ; 19,0-37,0%)	1,8 $10^9/L$	15,1%
MO (N 0,0-0,8 $10^9/L$ ; 3,0-11,0%)	0,7 $10^9/L$	6,1%
E (N 0,0-0,6 $10^9/L$ ; 0,5-5,0%)	0,1 $10^9/L$	0,5%
BA (N 0,0-0,2 $10^9/L$ ; 0,0-1,0%)	0,1 $10^9/L$	0,5%

RBC (N 3,9-5,0 $10^{12}/L$ )	4,39 $10^{12}/L$
Hb (N 120-160 g/L)	146 g/L
HCT (N 36,0-48,0%)	41,7 %
MCV (N 80-100 fL)	95,0 fL
MCH (N 28-36 pg)	33,3 pg
MCHC (N 310-370 g/L)	350 g/L

RDW-CV (N 10,0-16,5%CV)	14,0 %CV
PLT (N180-320 $10^9/L$ )	172 $10^9/L$
PCT (N 0,1-1,0%)	0,14 %
MPV (N 5,0-10,0 fl)	8,3 fL
PDW (N 12,0-18,0%)	16,9 %

**Conclusion:** Neutrophilic leucosytosis due to aseptic inflammatory response to the heart muscle necrosis

## Urine analysis

28.08.2015

<b>Colour</b>	<b>Light yellow</b>
<b>Specific gravity</b> (N 1,001-1,040)	<b>1,020</b>
<b>pH</b> (N 5,0-7,0)	<b>5.0</b>
<b>Protein</b> (N absent)	<b>0.068 g/L</b>
<b>Glucose</b> (N absent)	<b>Absent</b>
<b>Eritrocytes</b> (N single)	<b>1-2/HPF</b>
<b>Leucocytes</b> (N 6-8 in field)	<b>8-10/HPF</b>
<b>Transitional epithelium</b> (N single)	<b>sometimes</b>
<b>Tubular epithelium</b> (N single)	<b>single</b>
<b>Hyaline casts</b> (N single)	<b>0-1/HPF</b>
<b>Granular casts</b> (N absent)	<b>single</b>
<b>Crystals of oxalate</b> (N absent)	<b>sometimes</b>
<b>Crystals of uric acid</b> (N absent)	<b>sometimes</b>

**Conclusion:** Mild proteinuria, less than 1,0 g/L; proteinuria is associated with an increased risk of mortality from acute MI

## Biochemical blood profile

Glucose (3.9 - 6.4 venous blood) — 4.1 mmol/L

	28.08.2015
<b>Bilirubin Total</b> (N 17 - 21 mkmol/L)	<b>9,98</b> <b>mkmol/L</b>
<b>Bilirubin Direct</b> (N 0 - 7,9 mkmol/L)	<b>4,85</b> <b>mkmol/L</b>
<b>Bilirubin Indirect</b> (N < 19 mkmol/L)	<b>5,13</b> <b>mkmol/L</b>
<b>ALT</b> (N < 41 U/L)	<b>64,2 U/L</b>
<b>AST</b> (N < 35 U/L)	<b>103,4 U/L</b>

	28.08.2015
<b>Creatinine</b> (N 62-106 mkmol/L)	<b>424,97</b> <b>mkmol/L</b>
<b>Urea</b> (N 3,0-9,2 mmol/L)	<b>32,6</b> <b>mmol/L</b>
<b>Potassium</b> (N 3,5-5,1 mmol/L)	<b>3,1</b> <b>mmol/L</b>
<b>Total Protein</b> (N 66-83 g/L)	<b>48,4 g/L</b>

- **Conclusion:** Hyperfermentemia (increased levels of AST, ALT) may be due to either acute MI and liver dysfunction. Raised levels of cretinine, urea – signs of renal insufficiency. Hypokaliemia increases incidence of malignant ventricular arrhythmias . Hypoproteinemia may be due to either proteinuria and liver dysfunction.

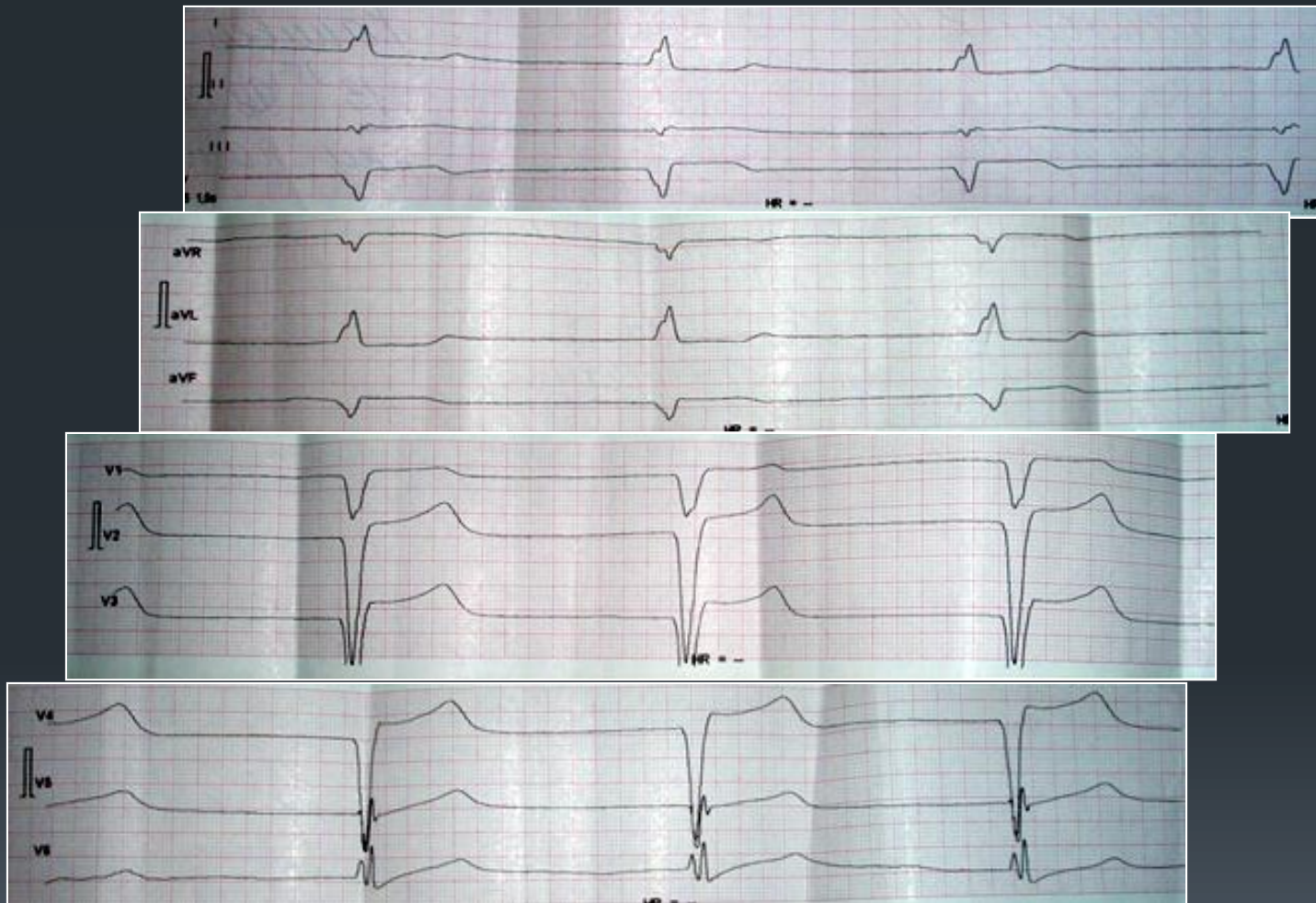
## Cardiac Biomarkers

	28.09.2015		29.09.2015	
	13:30	19:00	01:00	7:00
<b>CK-NAC</b> (N < 190 U/l)	<b>364.3 U/l</b>	<b>297.7 U/l</b>	<b>255.7 U/l</b>	<b>210.1 U/l</b>
<b>CK-MB</b> (N 0-24 U/l)	<b>68.83 U/l</b>	<b>63.8 U/l</b>	<b>44.48 U/l</b>	<b>33.72 U/l</b>
<b>Troponin I</b> (N < 0.01 ng/ml )	<b>12.94 ng/ml</b>			

**Conclusion:** Increased levels of cardiac enzymes (sign of damage to the cardiomyocytes)

# Clinical data

5



**ECG:** Bradycardia, heart rate 40 bpm, junctional rhythm, LBBB (QRS 0.14 ms), acute circular MI (ST elevation > 2 mm III, AVF, V1-V5, ST depression 1 mm I, AVL).

## TRANSTHORACIC ECHOCARDIOGRAM DATA 10.09.2015:

Sclerosis of aorta, aortic and mitral valve cusps. Mild valvular aortic stenosis. Aortic valve regurgitation I-II degree. Mitral valve regurgitation II degree. Tricuspid valve regurgitation I-II degree. Pulmonary artery valve regurgitation I degree. Dilation of all heart chambers. Left ventricle and right ventricle hypertrophy. Hypokinesia of the ventricular septa, basal and diaphragmal part of posterior wall, basal part of lateral wall of the left ventricle. Hydropericardium. Pulmonary hypertension I degree.

Ao D	(20 – 37 mm)	<b>39 mm</b>	EF	(55 – 78 %)	<b>46 %</b>
AvO	(17 – 26 mm)	<b>16 mm</b>	FS	(28 – 44 %)	<b>23 %</b>
LA	(<38mm)	<b>48 mm</b>	VST	(6-11mm)	<b>16,7 mm</b>
MvO	(28 mm)	<b>25 mm</b>	RAD		<b>57 mm</b>
LV EDD	(35 – 55 mm)	<b>56,3 mm</b>	RVD	(9 – 26 mm)	<b>45,0 mm</b>
LV ESD	(28 – 33 mm)	<b>43 mm</b>	RVAW	(3 – 6 mm)	<b>8 mm</b>
LVPW	(6 – 11 mm)	<b>16,3mm</b>			

**Conclusion:** Signs of total heart failure with hypertrophy and dilation of heart chambers, valvular regurgitation, LV contractility reduction. Development of pulmonary hypertension. Hydropericardium. Hypokinesia of the LV posterior wall, which is affected by infarction.

## ULTRASOUND ABDOMEN

Diffuse increase echogenic density of liver

Hepatomegaly:

- vertical oblique size of the right lobe **165 mm** (N<150 mm)
- left lobe thickness **110** (N<65 mm)

V. Cava distension, liver congestion

Diffuse changes of echogenic density of the pancreas with normal organ's size

Bilateral hydrothorax

**Conclusion:** diffuse alteration of liver and pancreatic parenchyma; hepatomegaly; liver congestion, bilateral hydrothorax – signs of total congestive heart failure

# Clinical Diagnosis

## MAIN DISEASE

Coronary artery disease. Acute repeated ST elevated circular myocardial infarction (28.08.2015). Postinfarction inferior wall cardiosclerosis (2006). Killip IV

Essential hypertension III stage, 2 grade

Persistent atrial fibrillation, tachysystolic form

Risk score 4 (very high).

Chronic congestive heart failure II NYHA with the reduction of LV contractility

## COMPLICATION

Junctional rhythm, bradycardia

Acute prerenal failure

## CONCOMINANT DIAGNOSIS

Alcoholic liver disease



# Course of Disease

1

30.08. 2015 7- 00

- **Complains:** black colored stools, fatigue, dizziness.
- **Inspection:**    **T** - 36,5 °C                      **Pulse** - 34 bpm  
                         **BP** - 90/60 mm Hg              **Respiratory rate** - 20 pm

Patient is drowsy and sluggish. Skin and mucous membranes are pale and dry.

Bronchial breath sounds of the lungs to auscultation. Decrease breath sounds in bases. Rhonchi and crackles are not auscultated.

Pulse is regular, soft and small (pulsus filiformis).

Soft S1 heart sound to auscultation.

Abdomen is soft and tender in epigastric region. Hepatomegaly (+4cm), palpation is tender. The kidneys are not palpable.

# Course of Disease

2

	28.08.2015		01.09.2015	
WBC (4,0-9,0 $10^9/L$ )	11,8 $10^9/L$		12,5 $10^9/L$	
NE (1,7-7,7 $10^9/L$ ; 47,0-72%)	9,1 $10^9/L$	77,8%	9,5 $10^9/L$	76,1%
LY (0,4-4,4 $10^9/L$ ; 19,0-37,0%)	1,8 $10^9/L$	15,1%	1,7 $10^9/L$	13,7%
MO (0,0-0,8 $10^9/L$ ; 3,0-11,0%)	0,7 $10^9/L$	6,1%	0,9 $10^9/L$	7,5%
E (0,0-0,6 $10^9/L$ ; 0,5-5,0%)	0,1 $10^9/L$	0,5%	0,3 $10^9/L$	2,2%
BA (0,0-0,2 $10^9/L$ ; 0,0-1,0%)	0,1 $10^9/L$	0,5%	0,1 $10^9/L$	0,5%
RBC (3,9-5,0 $10^{12}/L$ )	4,39 $10^{12}/L$		2,39 $10^{12}/L$	
Hb (120-160 g/L)	146 g/L		76 g/L	
HCT (36,0-48,0 %)	41,7%		23,1%	
MCV (80-100 fL)	95,0 fL		96,7 fL	
MCH (28-36 pg)	33,3 pg		31,8 Pg	
MCHC (310-370 g/L)	350 g/L		329 g/L	
RDW-CV (10,0-16,5%CV)	14,0%CV		13,7 %CL	
PLT (180-320 $10^9/L$ )	172 $10^9/L$		183 $10^9/L$	
PCT (0,1-1,0%)	0,14%		0,14 %	
MPV (5,0-10,0 fL)	8,3 fL		7,9 fL	
PDW (12,0-18,0%)	16,9%		18,1%	

**Conclusion:**  
rapid significant  
decline level of  
RBC, Hb, and  
HCT – support  
acute GIT  
bleeding; minor  
increase level of  
leukocytes.

## FIBROSCOPY:

- Esophagus is normal.
- Clear fluid present in the stomach. Mucosa is pale-pink.
- Several acute ulcers 0,5-0,8 cm in diameter, covered by fibrin is localized in antrum.
- Pylorus and duodenum are normal.

**Conclusion:** Acute stomach ulcers.

30.08. 2015 19-00

At patient took place respiratory arrest and cardiac arrest:  
absence of the breathing and pulsation of the main arteries,  
pupil dilation, loss of consciousness

ECG-monitor: isoline

At patient developed CLINICAL DEATH

Emergency measures:

- IV epinephrine
- Indirect cardiac massage

After 3 min emergency measures were successful: cardiac  
activity and respiration were restored

PS 80 bpm

BP 150/100 mmHg

RR 18 per min

# Course of Disease

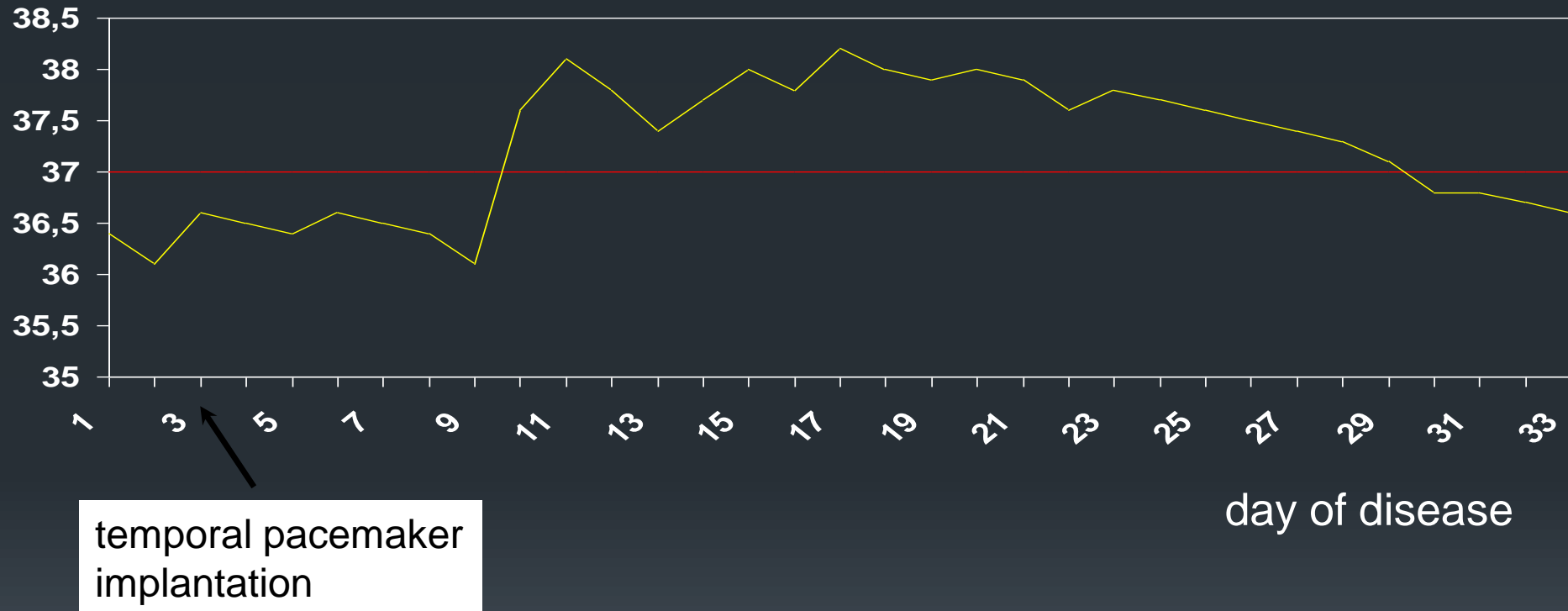
5

- In this case the most prominent atherosclerotic plaque is localized in the right coronary artery. The right coronary artery distributes blood to right ventricle, right atrium, posterior portion of the interventricular septum, posterior wall of the left ventricle and the heart conduction system (including sinoatrial *node*). Ischemia of SA node may lead to its dysfunction (bradycardia, SA arrest, etc.)
- Considering the severity of the patient's condition, refractory bradycardia (HR 40 bpm), developed acute renal failure and GIT bleeding, clinical death, 31.08.09, to improve patient's condition, temporal pacemaker was implanted

# Course of Disease

6

## DYNAMIC OF BODY TEMPERATURE



**Conclusion:** seven days after pacemaker implantation fever has been developed; temporal pacemaker implantation is predisposing factor to infection and infective endocarditis

## DYNAMIC OF COMPLETE BLOOD COUNT

	03.09.2015		04.09.2015		25.09.2015	
WBC (4,0-9,0 $10^9/L$ )	10,0 $10^9/L$		12,2 $10^9/L$		12,7 $10^9/L$	
NE (1,7-7,7 $10^9/L$ ; 47,0-72%)	6,8 $10^9/L$	69,2 %	10,2 $10^9/L$	84,1%	10,6 $10^9/L$	83,3 %
LY (0,4-4,4 $10^9/L$ ; 19,0-37,0%)	2,0 $10^9/L$	19,6 %	1,4 $10^9/L$	11,1%	1,3 $10^9/L$	10,6 %
MO (0,0-0,8 $10^9/L$ ; 3,0-11,0%)	0,7 $10^9/L$	6,8 %	0,4 $10^9/L$	2,9 %	0,5 $10^9/L$	3,6 %
E (0,0-0,6 $10^9/L$ ; 0,5-5,0%)	0,4 $10^9/L$	3,9 %	0,2 $10^9/L$	1,8 %	0,2 $10^9/L$	1,9 %
BA (0,0-0,2 $10^9/L$ ; 0,0-1,0%)	0,1 $10^9/L$	0,5 %	0,0 $10^9/L$	0,1 %	0,1 $10^9/L$	0,6 %
RBC (3,9-5,0 $10^{12}/L$ )	2,22 $10^{12}/L$		2.32 $10^{12}/L$		2,48 $10^{12}/L$	
Hb (120-160 g/L)	70 g/L		73 g/L		76 g/L	
HCT (36,0-48,0 )	21,3 %		22,1 %		23,4 %	
PLT (180-320 $10^9/L$ )	184 $10^9/L$		134 $10^9/L$		102 $10^9/L$	
ESR (1-10 mm/h)	7 mm/h		16 mm/h		7 mm/h	

## DYNAMIC OF COMPLETE BLOOD COUNT

	10.09.2015		14.09.2015		25.09.2015	
WBC (4,0-9,0 $10^9/L$ )	10,0 $10^9/L$		6,9 $10^9/L$		9,6 $10^9/L$	
NE (1,7-7,7 $10^9/L$ ; 47,0-72%)	7,9 $10^9/L$	79,7 %	5,3 $10^9/L$	75,8%	8,2 $10^9/L$	84,4 %
LY (0,4-4,4 $10^9/L$ ; 19,0-37,0%)	1,3 $10^9/L$	13,2 %	1,0 $10^9/L$	14,1%	1,1 $10^9/L$	11,8 %
MO (0,0-0,8 $10^9/L$ ; 3,0-11,0%)	0,5 $10^9/L$	4,7 %	0,3 $10^9/L$	4,7 %	0,3 $10^9/L$	3,4 %
E (0,0-0,6 $10^9/L$ ; 0,5-5,0%)	0,2 $10^9/L$	1,7 %	0,3 $10^9/L$	4,8 %	0,0 $10^9/L$	0,2 %
BA (0,0-0,2 $10^9/L$ ; 0,0-1,0%)	0,1 $10^9/L$	0,7 %	0,0 $10^9/L$	0,6 %	0,0 $10^9/L$	0,2 %
RBC (3,9-5,0 $10^{12}/L$ )	2,49 $10^{12}/L$		2.57 $10^{12}/L$		3,26 $10^{12}/L$	
Hb (120-160 g/L)	77 g/L		79 g/L		98 g/L	
HCT (36,0-48,0 )	23,6 %		24,2 %		29,5 %	
PLT (180-320 $10^9/L$ )	111 $10^9/L$		119 $10^9/L$		465 $10^9/L$	
ESR (1-10 mm/h)	18 mm/h		20 mm/h		13 mm/h	

**Conclusion:** persistent neutrophilic leukocytosis, increased ESR – signs of inflammation; positive trend of anemia: rising amount of RBC, hemoglobin and hematocrit



# Course of Disease

9

## TRANSTHORACIC ECHOCARDIOGRAM DATA 22.09.2015:

Sclerosis of aorta, aortic and mitral valve cusps. Aortic valve regurgitation II degree. Mild valvular aortic stenosis. Pathologic formation of the right coronary cusp of aortic valve (bacterial vegetation?). Mitral valve regurgitation II-III degree. Tricuspid valve regurgitation III degree. Pulmonary artery valve regurgitation I degree. Dilation of all heart chambers. Hydropericardium. Left ventricle and right ventricle hypertrophy. Hypokinesia of the ventricular septa, basal and diaphragmal part of posterior wall, basal part of lateral wall of the left ventricle. Pulmonary hypertension II degree.

Ao D	(20 – 37 mm)	<b>35,1 mm</b>	EF	(55 – 78 %)	<b>46 %</b>
AvO	(17 – 26 mm)	<b>16,6 mm</b>	FS	(28 – 44 %)	<b>21 %</b>
LA	(<38mm)	<b>53,2 mm</b>	VST	(6-11mm)	<b>16,4 mm</b>
MvO	(28 mm)	<b>29,4 mm</b>	RAD		<b>60,1 mm</b>
LV EDD	(35 – 55 mm)	<b>55,4 mm</b>	RVD	(9 – 26 mm)	<b>45 mm</b>
LV ESD	(28 – 33 mm)	<b>44,5 mm</b>	RVAW	(3 – 6 mm)	<b>7,9 mm</b>
LVPW	(6 – 11 mm)	<b>16,8 mm</b>			

**Conclusion:** Signs of total heart failure with hypertrophy and dilation of heart chambers, valvular regurgitation, LV contractility reduction. Development of pulmonary hypertension. Hydropericardium. Hypokinesia of the LV posterior wall, which is affected by infarction. The right coronary cusp of aortic valve bacterial vegetations.

- **CHEST X-Ray 22.09.2015:**

Focal and infiltrative is not observed

Pulmonary venous hypertension

In both pleural cavities occurs minor amount of fluid

Heart is enlarged, left border is harshly displaced to the left

Aortic arch sclerosis

**Conclusion:** Pulmonary congestion. Bilateral hydrothorax. Cardiomegaly.

Absence of pulmonary seeding

## BLOOD CULTURE

- 23.09.2016 # 134 – negative
- 25.09.2016 # 136 – negative

**Conclusion:** culture negative infective endocarditis may be due to prior administration of antibiotics

## DYNAMIC OF CARDIAC BIOMARKERS

	28.09.2015		29.09.2015	
	13:30	19:00	01:00	7:00
<b>CK-NAC</b> (N < 190 U/l)	<b>364.3 U/l</b>	<b>297.7 U/l</b>	<b>255.7 U/l</b>	<b>210.1 U/l</b>
<b>CK-MB</b> (N 0-24 U/l)	<b>68.83 U/l</b>	<b>63.8 U/l</b>	<b>44.48 U/l</b>	<b>33.72 U/l</b>
<b>Troponin I</b> (N < 0.01 ng/ml )	<b>12.94 ng/ml</b>			

	30.08.2015	31.08.2015
<b>CK-NAC</b> (N < 190 U/l)	125.5 U/l	120.9 U/l
<b>CK-MB</b> (N 0-24 U/l)	<b>31.1 U/l</b>	<b>29.24 U/l</b>

**Conclusion:** dynamic is positive, gradual decline of cardiac enzymes prove stabilization of heart necrosis

## DINAMIC OF BIOCHEMICAL PROFILE

	28.08. 2015	30.08. 2015	02.09. 2015	03.09. 2015	07.09. 2015	09.09. 2015	10.09. 2015	14.09. 2015
<b>Creatinine</b> (N 62-106 mkmol/L)	424,97 mkmol/L	494,1 mkmol/L	120,99 mkmol/L	107,0 mkmol/L	116,0 mkmol/L	111,9 mkmol/ L	94,61 mkmol/L	74,77 mkmol/L
<b>Urea</b> (N 3,0-9,2 mmol/L)	32,6 mmol/L		28,4 mmol/L		9,0 mmol/L		9,8 mmol/L	
<b>Potassium</b> (N 3,5-5,1 mmol/L)	3,1 mmol/L	5,0 mmol/L						
<b>Total protein</b> (N 66-83 g/L)		48,4 g/L		45,2 g/L	49,0 g/L		50,1 g/L	

**Conclusion:** positive dynamic in urea and creatinine, temporal pacemaker implantation improves kidney perfusion, acute renal failure abates; also occurs raising of total protein level; raising level of potassium: hypokalemia in the early phase of an acute MI is activation of the sympathetic nervous system leading to an influx of potassium from the extracellular to the intracellular body fluid compartment.

## DYNAMIC OF LIVER TESTS AND ENZYMES

	28.08.2015	02.09.2015	03.09.2015	07.09.2015	10.09.2015
<b>Bilirubin Total</b> (N 17 - 21 mkmol/L)	9,98 mkmol/L	13,53 mkmol/L	12,11 mkmol/L	14,87 mkmol/L	9,9 mkmol/L
<b>Bilirubin Direct</b> (N 0 - 7,9 mkmol/L)	4,85 mkmol/L				
<b>Bilirubin Indirect</b> (N < 19 mkmol/L)	5,13 mkmol/L				
<b>ALT</b> (N < 41 U/L)	64,2 U/L	74,64 U/L	80,0 U/L	46,6 U/L	31,4 U/L
<b>AST</b> (N < 35 U/L)	103,4 U/L	96,5 U/L	56,0 U/L	42,5 U/L	29,2 U/L

**Conclusion:** positive dynamic in ASL and ALT levels, gradual decline  
further normalization

an

# Clinical syndromes



- ACUTE CORONARY SYNDROME
- ARTERIAL HYPERTENSION
- HEART FAILURE
- JUNCTIONAL RHYTHM
- INFLAMMATION
- RENAL FAILURE
- GASTROINTESTINAL BLEEDING
- HYPOPROTEINEMIA
- ANEMIA
- INFECTIVE ENDOCARDITIS
- OBESITY

# Clinical syndromes classification

1

## ACUTE CORONARY SYNDROME

- Unstable angina
- ST segment elevation myocardial infarction (STEMI)
- Non ST segment elevation myocardial infarction (NSTEMI)



# Clinical syndromes classification

2

## ARTERIAL HYPERTENSION (I)

- **Essential**

- **Secondary:**

- I. Renal diseases

- II. Endocrine hypertension

- III. Hemodynamic hypertension:

- IV. Neurogenic hypertension:

- V. Special forms:

# Clinical syndromes classification

3

## ARTERIAL HYPERTENSION (III)

Category	Systolic		Diastolic
Optimal	< 120	and	< 80
Normal	120–129	and/or	80–84
High normal	130–139	and/or	85–89
Grade 1 hypertension	140–159	and/or	90–99
Grade 2 hypertension	160–179	and/or	100–109
Grade 3 hypertension	≥ 180	and/or	≥ 110
Isolated systolic hypertension	≥ 140	and	< 90

# Clinical syndromes classification

4

## HEART FAILURE

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- Acute heart failure
- Chronic heart failure
- Left sided heart failure
- Right sided heart failure
- Total heart failure
- Heart failure with systolic dysfunction of left ventricle (EF less than 40%)
- Heart failure with diastolic dysfunction of left ventricle (EF more than 50%)
- Heart failure with preserved function of left ventricle (EF more than 50%)

# Clinical syndromes classification

5

## **ACUTE HEART FAILURE**

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### **KILLIP CLASSIFICATION**

**CLASS I:** No evidence of heart failure (mortality 6%)

**CLASS II:** Findings of mild to moderate heart failure (S3 gallop, rales less than half-way up lung fields or elevated jugular venous pressure (mortality 17%)

**CLASS III:** Pulmonary edema (mortality 38%)

**CLASS IV:** Cardiogenic shock defined as systolic blood pressure < 90 and signs of hypoperfusion such as oliguria, cyanosis, and sweating (mortality 67%)

# Clinical syndromes classification

6

## CHRONIC HEART FAILURE

### Classification of Heart Failure: ACC/AHA Stage vs NYHA Class

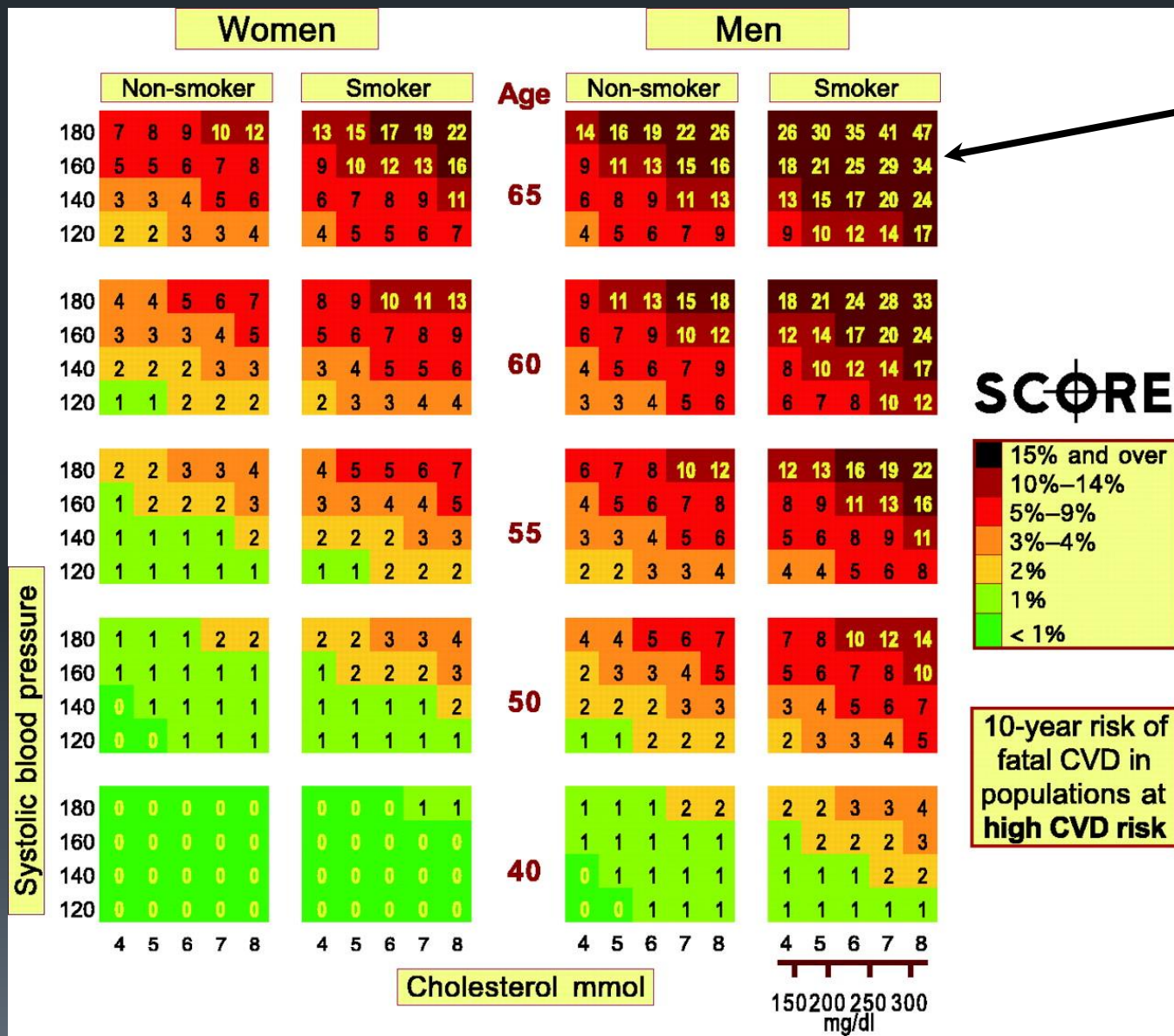
ACC/AHA Heart Failure Stage	NYHA Functional Class
A. At risk for heart failure but without structural heart disease or symptoms	None
B. Structural heart disease but without heart failure	I. Asymptomatic
C. Structural heart disease with prior or current heart failure symptoms	II. Symptomatic with moderate exertion III. Symptomatic with minimal exertion
D. Refractory heart failure requiring specialized interventions	IV. Symptomatic at rest

Hunt SA et al. *Circulation*. 2001;104:2996-3007.

Farrell MH et al. *JAMA*. 2002;287:890-897.

# Clinical syndromes classification

7



Patient:

- 65 years old
- smoker
- BP 160/90 mmHg

Score 4 very high



# Clinical syndromes classification

8

## JUNCTIONAL RHYTHM

- P wave precede the QRS complex
- P wave follow the QRS complex
- P wave occur during the QRS complex



# Clinical syndromes classification

9

## INFLAMMATION

---

### Classification according to duration

- Per-acute inflammation
- Acute inflammation
- Sub-acute inflammation
- Chronic inflammation

### Classification according to principle constituent of exudates

- Serous
- Catarrhal
- Fibrinous
- Suppurative (purulent)
- Hemorrhagic
- Lymphocytic

### Classification according to etiology

- Biological inflammation
- Chemical
- Physical
- Immune factors
- Classification according to location
- Localized
- Widespread or Systemic



# Clinical syndromes classification

10

## RENAL FAILURE (I)

### ■ ACUTE:

- Prerenal
- Intriniscic
- Obstructive

Grade	Glomerular filtration rate (GFR) criteria	Urine output (UO) criteria
R, Risk	Serum creatinine increase:1.5-fold; GFR decrease > 25%	UO < 0.5 ml/kg/ h for 6 h
I, Injury	Serum creatinine increase:2-fold; GFR decrease > 50%	UO < 0.5 ml/kg/ h for 12 h
F, Failure	Serum creatinine increase:3- fold; GFR decrease > 75%; serum creatinine decrease > 350umol/ liter(4 mg/dl) with acute increase 44 umol/liter (0.5 mg/dl)	UO < 0.3 ml/kg/h for 24 h or anuria for 12 h
L, Loss	Persistent ARF = complete loss of renal function for > 4 weeks	
E, Endstage	End stage renal disease(ESRD) = complete loss of renal function for > 3 months	

# Clinical syndromes classification

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## RENAL FAILURE (II)

### ■ **CHRONIC:**

- **Stage 1:** Kidney damage with normal or increased GFR ( $>90$  mL/min/1.73 m<sup>2</sup>)
- **Stage 2:** Mild reduction in GFR (60-89 mL/min/1.73 m<sup>2</sup>)
- **Stage 3:** Moderate reduction in GFR (30-59 mL/min/1.73 m<sup>2</sup>)
- **Stage 4:** Severe reduction in GFR (15-29 mL/min/1.73 m<sup>2</sup>)
- **Stage 5:** Kidney failure (GFR  $< 15$  mL/min/1.73 m<sup>2</sup> or dialysis)

# Clinical syndromes classification

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## GASTROINTESTINAL BLEEDING (I)

- **According to localization:**

- Upper gastrointestinal bleeding
- Lower gastrointestinal bleeding

- **According to the duration:**

- Acute
- Chronic

- **According to functional state:**

- Compensated

- Decompensated

- **According to the intensity:**

- Profuse bleeding
- Occult bleeding

# Clinical syndromes classification

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## GASTROINTESTINAL BLEEDING (II)

### ETIOLOGY

- **Upper gastrointestinal bleeding**
  - Esophagitis and gastroesophageal reflux disease
  - Mallory-Weiss tear
  - Varices (portal hypertensive gastropathy)
  - Peptic ulcer
  - **Acute stress gastritis**
  - Cancer of the stomach
  - Angiodysplasia
- **Lower gastrointestinal bleeding**
  - Hemorrhoids
  - Cancer
  - Angiodysplasia
  - Ulcerative colitis
  - Crohn's disease
  - Angiodysplasia
  - Diverticulosis / diverticulitis

# Clinical syndromes classification

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## CYTOLYSIS

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### CAUSES

#### Liver diseases:

- Cirrhosis
- Viral hepatitis acute and chronic
- Nonalcoholic steatohepatitis
- **Alcoholic liver disease**
- Medications and toxins
- Autoimmune hepatitis
- Hemochromatosis
- Cystic fibrosis
- Wilson disease

#### Other causes:

- Celiac disease
- Hemolysis
- Myopathy
- Excessive physical
- Hyperthyrosis
- Metabolic syndrome
- Diabetes mellitus
- **Myocardial infarction**
- Ischemic heart disease
- Congestive heart failure

# Clinical syndromes classification

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## HYPOPROTEINEMIA

- **Protein-losing gastroenteropathy:** pancreatic dysfunction, bacterial overgrowth in the small intestine, gastrointestinal infection, parasite infestations, diarrhea, Crohn's disease, ulcerative colitis, small intestine resection, AIDS, cancer
- **Malnutrition**
  - **Liver disease**
  - **Renal disease** (nephrotic syndrome)
- **Sepsis**

# Clinical syndromes classification

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## ANEMIA

### ETHIOLOGIC CLASSIFICATION I. IMPAIRED PRODUCTION

- **Disturbance of proliferation and differentiation of stem cells**
  - Pure red cell aplasia
  - Aplastic anemia
  - Anemia of renal failure
  - Anemia of endocrine disorders
- **Other mechanisms of impaired RBC production**
  - Myelophthistic anemia
  - Myelodysplastic syndrome
  - Anemia of chronic inflammation
- **Disturbance of proliferation and maturation of erythroblasts**
  - Pernicious anemia
  - Vitamin B12 deficiency anemia
  - Anemia of folic acid deficiency
  - Anemia of prematurity
  - Iron deficiency anemia
  - Thalassemias
  - Congenital dyserythropoietic anemias
  - Anemia of renal failure

# Clinical syndromes classification

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## ANEMIA

### ETHIOLOGIC CLASSIFICATION

#### II. INCREASED DESTRUCTION (HEMOLYTIC ANEMIAS)

##### ■ **Intrinsic (intracorpuscular) abnormalities**

- Hereditary elliptocytosis
- Hereditary spherocytosis
- Abetalipoproteinemia
- Enzyme deficiencies
  - Pyruvate kinase and hexokinase deficiencies
  - Glucose-6-phosphate dehydrogenase deficiency and glutathione synthetase deficiency
- Hemoglobinopathies
  - Sickle cell anemia
  - Hemoglobinopathies causing unstable hemoglobins
- Paroxysmal nocturnal hemoglobinuria

##### ■ **Extrinsic (extracorpuscular) abnormalities**

- Antibody-mediated
  - Warm autoimmune hemolytic anemia
  - Cold agglutinin hemolytic anemia
  - Rh disease
  - Transfusion reaction to blood transfusions
- Mechanical trauma to red cells
  - Microangiopathic hemolytic anemias
  - intravascular coagulation
  - Infections, including malaria
  - Heart surgery
  - Haemodialysis



# Clinical syndromes classification

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## ANEMIA

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### III. BLOOD LOSS

- Anemia of prematurity
- Trauma or surgery
- **Gastrointestinal tract lesions**
- Gynecologic disturbances
- Infection by intestinal nematodes

### IV. FLUID OVERLOAD

- Excessive sodium or fluid intake, sodium or water retention and fluid shift into the intravascular space
- Anemia of pregnancy

# Clinical syndromes classification

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## ANEMIA

### HEMOGLOBIN LEVEL CLASSIFICATION

- Mild - Hb 90 – 120 g/L
- Moderate - Hb 70 – 90 g/L
- Severe - Hb <70 g/L

# Clinical syndromes classification

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## INFECTIVE ENDOCARDITIS

### Duration

- Acute bacterial endocarditis
- Subacute bacterial endocarditis

### Culture results

- Culture-positive
- Culture-negative

### Valve type

- Native-valve endocarditis
- Prosthetic-valve endocarditis

# Clinical syndromes classification

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## INFECTIVE ENDOCARDITIS

### IE according to localization of infection and presence or absence of intracardiac material

- Left-sided native valve IE
- Left-sided prosthetic valve IE (PVE)
  - Early PVE: < 1 year after valve surgery
  - Late PVE: > 1 year after valve surgery
- Right-sided IE
- Device-related IE (permanent pacemaker or cardioverter-defibrillator)

### IE according to the mode of acquisition<sup>22</sup>

- Health care-associated IE
  - Nosocomial: IE developing in a patient hospitalized > 48 hours prior to the onset of signs / symptoms consistent with IE
  - Non nosocomial: Signs and / or symptoms of IE starting < 48 hours after admission in a patient with health care contact defined as:
    - 1) home-based nursing or intravenous therapy, haemodialysis, or intravenous chemotherapy < 30 days before the onset of IE; or
    - 2) hospitalized in an acute care facility < 90 days before the onset of IE; or
    - 3) resident in a nursing home or long-term care facility
- Community-acquired IE Signs and / or symptoms of IE starting < 48 hours after admission in a patient not fulfilling the criteria for health care-associated infection
- Intravenous drug abuse-associated IE IE in an active injection drug user without alternative source of infection

## INFECTIVE ENDOCARDITIS

### Active IE

- IE with persistent fever and positive blood cultures *or*
- Active inflammatory morphology found at surgery *or*
- Patient still under antibiotic therapy *or*
- Histopathological evidence of active IE

### Recurrence

- Relapse: Repeat episodes of IE caused by the same microorganism < 6 months after the initial episode
- Reinfection: Infection with a different microorganism  
Repeat episode of IE caused by the same microorganism > 6 months after the initial episode

# Clinical syndromes classification

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## INFECTIVE ENDOCARDITIS

The Duke diagnostic criteria

Major blood culture criteria for IE include the following:

- Two blood cultures positive for organisms typically found in patients with IE
- Blood cultures persistently positive for one of these organisms, from cultures drawn more than 12 hours apart
- Three or more separate blood cultures drawn at least 1 hour apart

Major echocardiographic criteria include the following:

- **Echocardiogram positive for IE, documented by an oscillating intracardiac mass on a valve or on supporting structures, in the path of regurgitant jets, or on implanted material, in the absence of an alternative anatomic explanation**
- Myocardial abscess
- Development of partial dehiscence of a prosthetic valve
- New-onset valvular regurgitation

# Clinical syndromes classification

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## INFECTIVE ENDOCARDITIS

The Duke diagnostic criteria

Minor criteria for IE include the following:

- Predisposing heart condition or intravenous drug use
- Fever of 38°C (100.4°F) or higher
- Vascular phenomenon, including major arterial emboli, septic pulmonary infarcts, mycotic aneurysm, intracranial hemorrhage, conjunctival hemorrhage, or Janeway lesions
- Immunologic phenomenon such as glomerulonephritis, Osler nodes, Roth spots, and rheumatoid factor
- Positive blood culture results not meeting major criteria or serologic evidence of active infection with an organism consistent with IE
- Echocardiogram results consistent with IE but not meeting major echocardiographic criteria

# Clinical syndromes classification

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## INFECTIVE ENDOCARDITIS

The Duke diagnostic criteria

### DEFINITE IE

- Pathologic criteria

Microorganism: demonstrated by culture or histology in a vegetation, or in a vegetation that has embolized, or in an intracardiac abaccess OR

Pathologic lesions: vegetation or intracardiac abaccess, confirmed by histology, showing active endocarditis

- Clinical criteria

2 major criteria OR

1 major criteria and 3 minor criteria OR

5 minor criteria

### POSSIBLE IE

Findings consisting with IE that fall short of “DEFINITE IE” but not rejected:

1 major criteria and 2 minor criteria OR

3 minor criteria

### REJECTED IE

Firm alternate diagnosis for manifestations of endocarditis OR

Resolution of manifestation of endocarditis, with antibiotic therapy for four days or less OR

No pathologic evidence of IE at a surgery or autopsy after antibiotic therapy for four days or less



## OBESITY

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BMI (kg/m <sup>2</sup> )		Classification
from	up to	
	18.5	underweight
18.5	25.0	Normal weight
25.0	30.0	overweight
30.0	35.0	class I obesity
35.0	40.0	class II obesity
40.0		class III obesity

# Final Diagnosis

## **MAIN DISEASE**

Coronary artery disease. Repeated ST elevated circular myocardial infarction (28.08.2015). Postinfarction inferior wall cardiosclerosis (2006). Killip IV

Essential hypertension III stage, 2 grade

Persistent atrial fibrillation, tachysystolic form

Chronic heart failure with systolic dysfunction of left ventricle

Risk score 4 (very high).

## **COMPLICATION**

Junctional rhythm, bradycardia (40 bpm)

Acute prerenal failure

Clinical death (30.08.2015)

Temporal pacemaker implantation (31.08.2015)

Possible nasocomial active device-related (temporal pacemaker) infective endocarditis

Acute gastric stress ulcers, GIT bleeding

Anemia of blood loss, moderate

## **CONCOMINANT DIAGNOSIS**

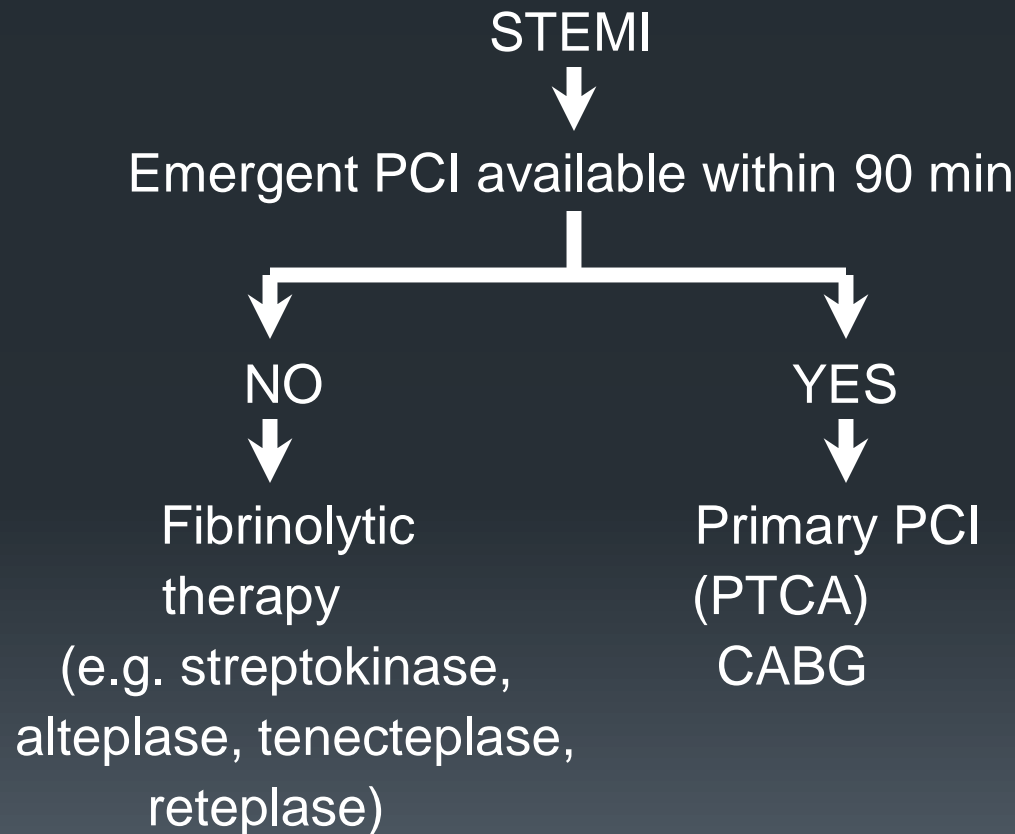
Alcoholic liver disease, alcoholic hepatitis

Obesity class I

## **LIFESTYLE MODIFICATION:**

- Diet: low in saturated fat and high in omega-3 fat, low carbohydrates, low sodium (3g/d)
- Limit alcohol consumption
- Body weight control: goal BMI 18,5 - 24,99
- Smoking cessation

## ACUTE CORONARY SYNDROME MANAGEMENT



# 3

B-blocker  
Nitrates  
Ca channel blocker (verapamil, diltiazem, nifedipine, amlodipine, felodipine)

# Management

4

## ACUTE CORONARY SYNDROME MANAGEMENT

**Adjunctive therapy:**      Statin (lovastatin, simvastatin, pravastatin, fluvastatin, atorvastatin, rosuvastatin )

   ACE inhibitor (captopril, ramipril, enalapril, quinapril, perindopril, lisinopril, benazepril)

   Aldosterone blockers (spironolactone, eplerenone)

   Proton pump inhibitor (omeprazole)

**Rehabilitation**

**Education**

**Counseling**

## **ACUTE CORONARY SYNDROME TREATMENT**

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- IV Morphine sulfate
- Low molecular weight heparin (enoxaparinum 80 mg/2d)
- Aspirin 75 mg/d
- Clopidogrel 75 mg/d
- Otorvastatin 80 mg/d
- Eplerenonum 25 mg/d
- Ramipril 2,5 mg/d
- Pantoprasol 40 mg/d

## **ADDITIONALLY**

- Thrombolytic therapy (IV Alteplase)      or
- PCI

## **BRADYCARDIA TREATMENT**

Atropine IV/IM

Transcutaneous pacing



## **BRADYCARDIA TREATMENT**

- Refractory bradycardia requires implantation of temporal pacemaker

## INFECTIVE ENDOCARDITIS TREATMENT

Recommendations: IE on pacemakers and implantable defibrillators	Class <sup>a</sup>	Level <sup>b</sup>
<b>A - PRINCIPLES OF TREATMENT:</b>		
Prolonged antibiotic therapy and device removal are recommended in definite CDRIE	I	B
Device removal should be considered when CDRIE is suspected on the basis of occult infection without other apparent source of infection	IIa	C
In patients with native or prosthetic valve endocarditis and an intracardiac device with no evidence of associated device infection, device extraction may be considered	IIb	C
<b>B - MODE OF DEVICE REMOVAL:</b>		
Percutaneous extraction is recommended in most patients with CDRIE, even those with large (> 10 mm) vegetations	I	B
Surgical extraction should be considered if percutaneous extraction is incomplete or impossible or when there is associated severe destructive tricuspid IE	IIa	C
Surgical extraction may be considered in patients with very large (> 25 mm) vegetations	IIb	C
<b>C – REIMPLANTATION:</b>		
After device extraction, reassessment of the need for reimplantation is recommended	I	B
When indicated, reimplantation should be postponed if possible to allow a few days or weeks of antibiotic therapy	IIa	B
Temporary pacing is not recommended	III	C
<b>D - PROPHYLAXIS</b>		
Routine antibiotic prophylaxis is recommended before device implantation	I	B

## INFECTIVE ENDOCARDITIS TREATMENT

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Staphylococci are the most frequent pathogens, *S. aureus* being predominant in the acute forms of pacemakers infection.

(Klug D, Lacroix D, Savoye C, Goullard L, Grandmougin D, Hennequin JL, Kacet S, Lekieffre J. Systemic infection related to endocarditis on pacemaker leads: clinical presentation and management. *Circulation* 1997;**95**:2098-2107. )

- Pacemaker removal
- IV Cefepime 1,0 g 2 times/d      14 days
- IV Vancomycin 2 g 1 times/d      7 days

## RENAL FAILURE TREATMENT

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- Supportive treatment

Measures to correct underlying causes of acute kidney injury

Maintenance of volume homeostasis and correction of biochemical abnormalities remain the primary goals of treatment and may include the following measures:

- Correction of fluid overload with furosemide
- Correction of severe acidosis with bicarbonate administration, which can be important as a bridge to dialysis
- Correction of hyperkalemia

- Renal replacement therapy

## **RENAL FAILURE TREATMENT**

- Reduction of cardiac output due to myocardial infarction and bradycardia due to sinoatrial ischemia, hypovolemia due to GI bleeding lead to prerenal acute kidney injury
- Temporal pacemaker implantation improves cardiac output and kidney perfusion
- IV solutions to increase blood volume is not indicated, because of increasing heart preload and heart demands

## GASTROINTESTINAL BLEEDING TREATMENT

- Therapeutic endoscopy (injection of epinephrine or sclerosants, electrocoagulation, application of hemoclips or endoclips, etc.)
- Proton pump inhibitors ( omeprazole, pantoprazole and esomeprazole)
- Histamine receptor antagonists (famotidine)
- Surgical treatment

## GASTROINTESTINAL BLEEDING TREATMENT

### ■ **Withhold:**

Low molecular weight heparin  
Aspirin  
Clopidogrel.

### ■ **Prescribe:**

#### — Hemostatic therapy:

IV  $\epsilon$ -aminocaproic acid 100,0 2 times/d

IV Etamsilate 12,5% 4,0 3 times/d

IV Menadione 1% 1,0 3 times/d

#### — Antisecretory drugs

IV Famotidine 20 mg 2 times/d

IV Pantoprazole 40 mg 1 time/d

## **ANEMIA TREATMENT**

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- Transfusion of packed red blood cells
- Iron supplementation (ferrous sulfate )
- Diet



## **ANEMIA TREATMENT**

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- Ferrous sulfate - 60 mg/d per os for 3 months
- Folic acid 400 mcg/day per os for 3 months

## ALCOHOLIC LIVER DISEASE TREATMENT

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- Essential phospholipids - 300 mg/ 2d for 3 months
- Argininum - 1,5 g/ 2d for 3 months

# Prognosis



- **Prognosis for recovery:**

Unfavorable

- **Prognosis for life:**

At this stage of disease take place irreversible structural heart changes (post MI heart remodeling and heart chamber dilation). At the present time main goal of the therapy is prevention further progression of congestive heart failure and improvement patient's life quality.

# Finale



- We illustrate a case report that demonstrates complicated myocardial infarction and remind clinicians that prompt recognition and management are critical in this uncommon grave clinical case
- Clinicians must keep potentially lethal complications in mind when evaluating these unstable patients
- Early, aggressive, and judicious treatment of these complications may substantially decrease the morbidity and mortality associated with acute myocardial infarction